- ***Biochemistry***
- ***L– 6***

Lipids Metabolism

Digestion :

Is the mechanical and chemical breakdown of food into smaller components that are more easily absorbed into a blood stream.

Main dietary lipids are :

Triglycerides

Phospholipids

Cholesterol

In the mouth :

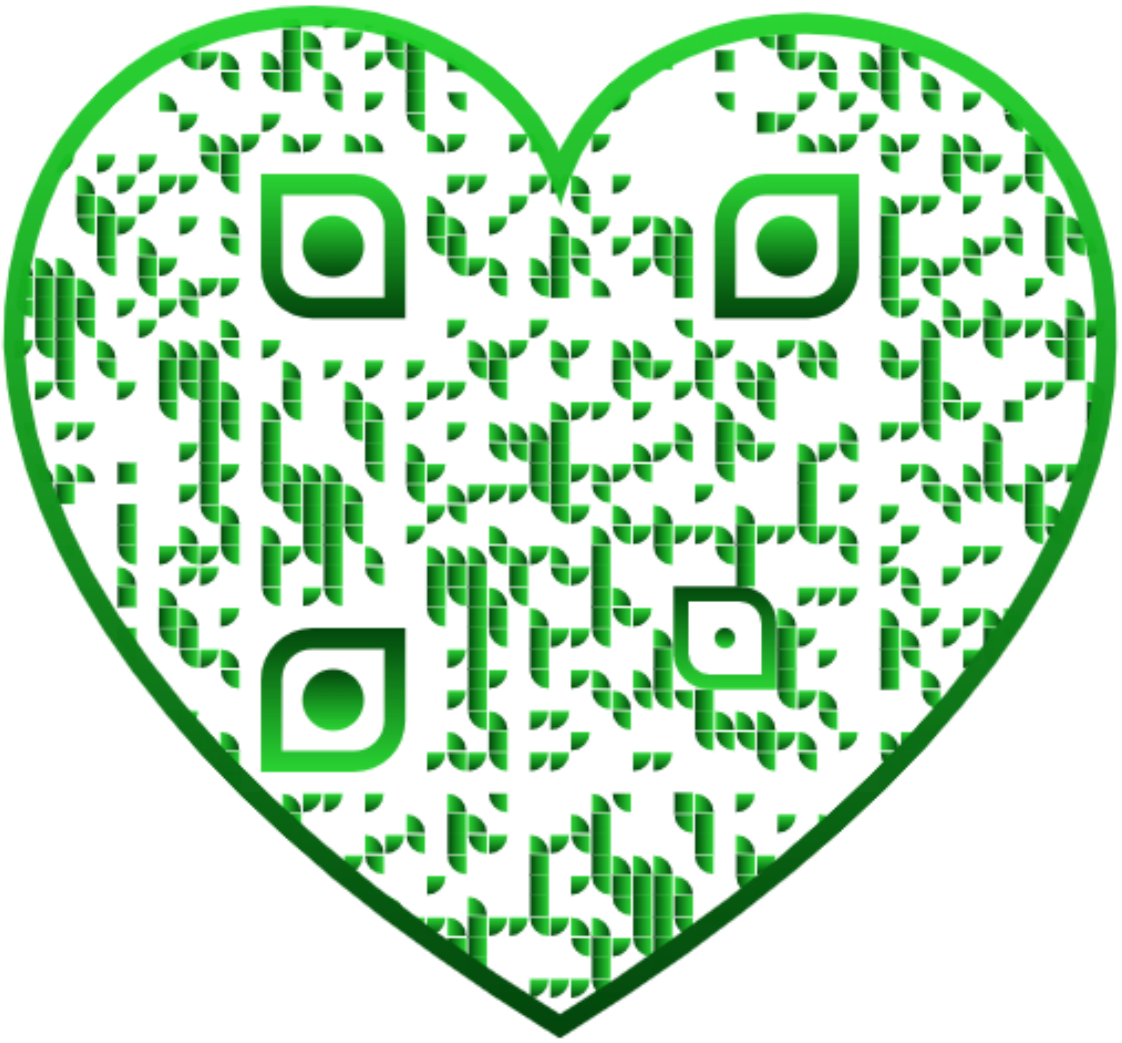
- The *lingual lipase* is specific for the ester bond number ***3*** of triglycerides.
- The digestion by *lingual lipase* is limited ؟؟ ليييييش
  Because the food remains short time in the mouth and lipids have low solubility.

Triglycerides —(**Lingual lipase**)→ *Fatty acids +1,2 diacylglycerol*

In the stomach :

- The *gastric lipase* is secreted Has the same activity of lingual lipase.

Triglycerides —(**Gastric lipase**)→ *fatty acids + 1,2 diacylglycerol*



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In the small intestine :

- Major site of lipid digestion. ؟؟ ليييييش
  Because the lipids are emulsified by bile salts ؟؟كيييف
  Emulsification is broken down of large lipid particles into small particles.

- *Pancreatic lipase* is specific for bond 1,3
- *Isomerase* transfer the fatty acids from ester number 2 to ester number 1.

pancreatic lipase علشان يتكسر بواسطة

Triglycerides —(*Pancreatic lipase*, *Colipase*, *Bile salts*, *Isomerase*)→ *Fatty acids + glycerol* (*isomerase* مع) / *Or* / *Fatty acids + 2-monoacylglycerol* (*isomerase* بدون)

Digestion of cholesterol Ester and Phospholipids :

1. *Cholesterol ester* —(*Cholesterol esterase*)→ *cholesterol + one fatty acid*

2. *Phospholipids* —(*Phospholipase A2*)→ *fatty acid + lysophospholipid*

- **المساعد هنا في الثنتين المعادلات يكون *bile salts***

- ***Biochemistry***
- ***L– 6***

Absorption of lipids :

1. Monoacylglycerol , Long-chine free fatty acid , Cholesterol, Lysophospholipids , Fat soluble vitamins and Bile salts are *combine* together to form ***micelles***.
2. A *micelles* enter into intestine by *endocytosis*.
3. Than combined with B_{48}(*apolipoprotien*) to form *chylomicrons* which enter *lymphatic* vessels.

Lipoprotein lipase: is enzyme found in blood breakdown of bonds number 1,2,3 of triglycerides of chylomicron.

Need for its activity apoprotein C-*II*.

4. After hydrolysis of triglycerides, the chylomicrons are called *chylomicron remnants* which taken up by *liver by endocytosis*.

- الناتج النهائي من التكسير هو fatty acid and glycerol
- يتم امتصاص الدهون على شكل chylomicron تدخل ال lymphatic vessels
- الي يكسر ال chylomicron في الدم هو lipoprotein lipase
- Lipoprotein lipase يكسر معظم ال chylomicron و المتبقي منه يدخل الكبد

Fate of glycerol :

1. Produce glucose
2. Produce energy
3. Produce triglycerides

Fate of free fatty acids :

1. *Produce triglycerides.*
2. *Produce energy.*
3. *Odd fatty acids produce glucose.*
4. *Produce other compound e.g. prostaglandins.*

- ***Biochemistry***
- ***L– 6***

Lipolysis in adipose tissue :

Is breakdown of triglycerides into free fatty acids and glycerol. *What??*

In adipose tissue. *Where??*

During fasting, starvation or diabetes type 1. *When??*

Steps of lipolysis :

1. *Triglycerides* $\xrightarrow{\text{Hormone sensitive lipase}}$ *di or monoglycerides + fatty acid*

2. *Di or monoglycerides* $\xrightarrow{\text{Di or monoglyceride lipase}}$ *fatty acids + glycerol*

Regulation Of lipolysis by hormones :

1. During fasting Glucagon and adrenalin stimulate lipolysis.
2. *During feeding* insulin inhibits lipolysis.

Oxidation of fatty acids :

- **Fatty acids are oxidized in the body by different methods called :**
  1- *β-oxidation*
  2- *α-oxidation*
  3- *ω-oxidation*
  4- *oxidation of very long fatty acids in peroxisomes.*

- **The oxidation of fatty acid consist of three stage :**
  1- *Activation of fatty acids in the cytosol.*
  2- *Transport of activated fatty acid into mitochondria.*
  3- *β-oxidation of fatty acids in the mitochondria matrix.*

وصف العملية بمعادلة:

Free fatty acid + CoA + ATP —*Acyl-CoA Synthetase*→ acyl-CoA + AMP + PPI

هذه معناها حمض دهني مرتبط

*طبعًا في كل عملية "activation" يتم استهلاك 2ATP خليها معلومة في راسك عنحتاجها بعدين.

"Carnitine shuttle" هانا نكتفي باسم الناقل يلي هوه

acyl-CoA "المهم هذا الناقل ينقل لك ال
From outer mitochondria membrane into matrix of mitochondria.

β-oxidation : تعريف :

Its break down of fatty acids into Acetyl-CoA , $FADH_2$ AND $NADH+H^+$
in mitochondria and peroxisomes
every time
produce energy

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Steps of β-oxidation :

Step1 Free fatty acid + CoA + ATP —*Acyl-CoA dehydrogenase*→ Δ 2-trans-enoyl-CoA + FADH2 *oxidation*

Step2 2-trans-enoyl-CoA —*Enoyl-CoA hydratase*→ L-β-hydroxyacyl-CoA *Hydration* *Add H_2O*

Step3 L-β-hydroxyacyl-CoA + NAD^+ —*L -hydroxy Acyl-CoA dehydrogenase*→ β-keto acyl-CoA + $NADH+H^+$ *oxidation*

Step4 β-keto acyl-CoA —*Thiolase*→ fatty acyl-CoA *Cleavage*

Calculation of energy produced by stearic acid oxidation

IS 18 carbon fatty acid

IS 9 Acetyl-CoA *تقسم عدد الكربونات على 2 يطلع لك عدد ال "Acetyl-CoA"

IS 8 NADH

IS 8 FADH2

من هذه المعطيات تقدر تحسب ال ATP :

9 × 12 = 108 → *كل واحد Acetyl-coA يعطي 12ATP

8 × 3 = 24 → *كل واحد NADH يعطي 3ATP

8 × 2 = 16 → *كل واحد FADH2 يعطي 2ATP

نجمعهم ؟

148 =

148 -2 = **146**

ننقص 2ATP تذكر يلي قلت لك تحفظ انه نستخدم 2ATP ؟

*طبعًا في كل عملية "activation" يتم استهلاك 2ATP خليها معلومة في راسك عنحتاجها بعدين.

1- *Complete oxidation of palmitic acid (16) produce 129 ATP*
2- *Complete oxidation of palmitic CoA (16) produce 131 ATP*
3- *Complete oxidation of Arachidic acid (20) produce 163 ATP*

هانا ما طلع 129 رغم انه عدد الكربونات 16 السبب انه ما نقصنا ال2ATP لان موجود في أسم المركب "CoA"

Regulation of β-oxidation :

- During fasting glucagon and stimulates β-oxidation ***(catabolic)***
- During feeding insulin and inhibits β-oxidation ***(anabolic)***

Important of α-oxidation:

- Oxidation of branched chain fatty acid
- Formation of odd chain fatty acid
- Formation of α-hydroxy fatty acid

Importance of ω-oxidation:

- **(minor pathway for long and very long fatty acid)**

Done by :

Noor Aldeen Ahmed

&

Rugia Fateh

Ketone bodies : تعريف :

are water-soluble compounds
in mitochondria and liver
during fasting ,starvation and type1 of diabetes mellitus
produce energy

Ketogenesis : تعريف :

Production of ketone bodies
in mitochondria matrix in the liver
during fasting state ,starvation and type1 of diabetes mellitus
produce energy

Regulation of Ketogenesis :

- During fasting glucagon and adrenalin (epinephrine) stimulates Ketogenesis.
- During feeding insulin and inhibits Ketogenesis.
- During diabetes mellitus type1 insulin decrease or absent ,and glucagon stimulates Ketogenesis.

Steps of Ketogenesis :

Step1 2 acetyl-CoA —*Thiolase*→ Acetoacetyl-CoA

Step2 Acetoacetyl-CoA + acetyl-CoA —*HMG –CoA Synthase*→ 3-hydroxy-3-methylglutary-CoA (HMG-CoA)

Step3 HMG-CoA —*HMG –CoA lyase*→ Acetoacetate + acetyl-CoA

Step4 Acetoacetate + NAD^+ —*β –Hydroxy butyrate dehydrogenase*→ β-hydroxy butyrate +$NADH+H^+$

Step5 Acetone

ال "Acetoacetate" من تلقاء نفسه يحصله " decarboxylation "ويتحول إلى "acetone"

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☆*Ketolysis:*

- Breakdown of ketone, ***In*** extra hepatic tissue, ***During*** fasting & Starvation & Diabetic maleates Type 1 ***To*** produce energy.

- ***Steps of ketolysis:***

1. *B – Hydroxybutyrate* —(NAD^+, *B – Hydroxybutyrate dehydrogenase*)→ *acetoacetate* + $NADH + H^+$

2. *Acetoacetate* —(*Succinyl-CoA*; *B – Ketoacyl-CoA transferase*)→ *acetoacetyl-CoA* + *Succinate*

3. Acetoacetyl-CoA —(*Thiolase*)→ 2 Acetyl-CoA

- *B – Hydroxy butyric acid produce 27 ATP.*
- *Acetoacetic acid produce 24 ATP.*

Ketosis and ketoacidosis: ● سؤاااال اختباااار ركزووو

- Ketosis is accumulation of ketone bodies in blood with out any change in pH of blood.
- Ketoacidosis is excessive accumulation of ketone bodies in blood which lead to decrease blood pH.

Metabolism Of Lipoproteins

1. Metabolism of Chylomicron:

- The largest lipoproteins.
- Produced in small intestine.
- Transport dietary fat from intestine to the blood.
- Enriched by triglycerides.

Breakdown

- *Enters blood as Nascent Chylomicron B-48* غير ناضج(جاهز)
- *Added with ApoC & ApoE by HDL* خلاص الان يكون ناضج(جاهز)
- *Breakdown into glycerol & fatty acids & chylomicron remnants by lipoprotein lipase*
- *Chylomicron remnants enters liver by endocytosis*

Note/ lipoprotein lipase is enzyme found in blood breakdown of triglycerides. specific for bonds number 1&2&3.

2. Metabolism of (VLDL):

- Synthesized in liver.
- Transport endogenous lipids from liver to the extra hepatic tissue.
- Enriched by triglycerides.

Breakdown

1- Enters blood as nascent VLDL
2- Added with ApoC &ApoE by HDL
3- Breakdown into glycerol & fatty acids & IDL (VLDL remnants)
4- IDL (VLDL) has two ways
   - Enter liver. ApoE اذا كان مرتبط ب
   - Convert to LDL. ApoE اذا فقد

3. Metabolism of (LDL):

- Called bad cholesterol.
- Produce from VLDL.
- Transport cholesterol from liver to extra hepatic tissue.
- Resk factor for cardiovascular disease.
- Enriched in cholesterol.
- Increase in male.

4. Metabolism of (HDL):

- Good cholesterol
- Smallest lipoprotein
- Produce in liver & intestine
- Transport the cholesterol from extra hepatic tissue to the liver
- Reduce risk factor for cardiovascular diseases
- Sources of ApoC & ApoE

- *Enriched in cholesterol*
- *Increase in female*

كثير صح لكن سهلات قوي عكس ال LDL

سؤاااال/ ماهو افضل مكان لل cholesterol ؟؟ ولييييييش ؟؟

الكبد علشان انه :

1 . يتم طرد cholesterol الى العصاره الصفراويه
2 . يحول cholesterol الى bile acid
3 . يمنع تصنيع cholesterol

طيب سؤاااال ثاني / ما تأثير الدخان (السجائر) على cholesterol ؟؟

ترفع Cholesterol

و ال dietary fiber يثبط ال cholesterol

Cholesterol metabolism :

- *Synthesis in liver (50%).*
- *All tissue containing nucleated cell are able to synthesis cholesterol.*
- *Normal range (140-200 mg/dl).*
- *It's an essential structural component of cell membrane.*
- *Produce steroid protein.*

أهم إنزيم :
= Regulatory enzyme

↓

HMG-CoA reductase
(3-hydroxy-3-methylglutary-CoA)

Medical important :

- *Stain drugs*
- *Hypercholesterolemia*
- *Hypocholesterolemia*

Like : atorvastatin and lovastatin
Used to decrease cholesterol
هذه الأدوية تعطل عمل ال (HMG-CoA) ولمن يتعطل عمله يوقف صناعة الكولسترول.

CAUSES :

1. ***Familial hypercholesterolemia.***
2. ***Diabetes mellitus.***
3. ***Hypothyroidism.***
4. ***Obstructive jaundice.***
5. ***Nephrotic syndrome (Nephrosis).***
6. ***Increase dietary.***

CAUSES :

1. ***Liver cirrhosis.***
2. ***Haemolytic anemia.***
3. ***Hyperthyroidism.***

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****smoking = risk factor***
****dietary fibers = protect factor***
معلومات مهمة.

Protein metabolism :

Digestion of protein :

- **In the mouth :** ***No enzyme*** ***(mechanical reaction)***
- **In the stomach :** ***By two enzymes.***
- **In the intestine :** ***By nine enzymes.***

Pepsin : is secreted by the chief cell of the stomach as the ***inactive*** pepsinogen.
Activation of pepsinogen by (*HCL* and *Pepsin*).
Cleave peptide bonds ***adjacent*** to (*acidic amino acids* a*romatic amino acid*) ***or*** *branched*-chain amino acids ***or*** *methionine*.

Rennin : produced by gastric chief cells in ***infants*** to coagulate the milk protein.
Cutting peptide bond between *phenylalanine* **and** *methionine*.

Trypsin : produced by pancreas and secreted as the ***inactive*** trypsinogen
cleaved to form Trypsin by enzyme "***enteropeptidase***" .
cleaves peptide bonds adjacent to "*arginine*" "*lysine*"

Chymotrypsin : the chymotrypsinogen ***inactive*** zymogen is cleaved to form active Chymotrypsin by ***Trypsin***
cleaves peptide bonds adjacent to "*aromatic amino acid* "
"*leucine*" "*methionine*"

Elastase : the proelastase ***inactive*** zymogen is cleaved to form active Elastase by ***Trypsin***
cleaves peptide bonds adjacent to "*Alanine* " "*glycine*" "*serine*"

Carboxypeptidase A : ***inactive*** proCarboxypeptidase A
Converted to Carboxypeptidase A by ***Trypsin***
cleaves "*valine* " "*leucine*" "*isoleusine*" "Alanine" from the C-terminus.

Carboxypeptidase B : ***inactive*** proCarboxypeptidase B
Converted to Carboxypeptidase B by ***Trypsin***
cleaves "*Lysine* " "*arginine*" from the C-terminus.

Endopeptidase : cleaves peptide bonds adjacent to "*hydrophobic amino acid* " "*glycine*" "*serine*"

Aminopeptidases A : cleaving "*acidic amino acid* " from the N-terminus

Aminopeptidases N : cleaving "*neutral amino acid* " from the N-terminus

Dipeptidases *and* **tripeptidases** :produce amino acids from ***di*** and ***tri*** "***peptides***"

****all enzymes are Endopeptidase except Carboxypeptidase and Aminopeptidases are Exopeptidase.***
****Endopeptidase produce small peptides.***
****Exopeptidase produce small peptides and amino acids .***

معلومات مهمة.

Absorption of amino acids :

- *Absorbed from the intestinal lumen by the **sodium-amino acid transport system**.*
- *Amino acid transport from enterocytes to exterocytes by **amino acid side-chain**.*
- *Inter blood capillary by **simple diffusion.***

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By : Nooraldeen ahmed
Abdalrhman Naser

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Biochemistry
L → 10

☆ *Fat of amino acids :*

- *Synthesis of New proteins for growth or repair.*
- *Synthesis of many small nitrogen-containing compounds.*
- *Produce of energy ' glucose and triglycerides.*

☆ *General catabolic reaction of amino acids :*

1. ***Transmutation*** *(Aminotransferase) :*
   - *Transfer of an amino group from a −Amino acid To a −keto acid to produce new a −Amino acid and a −keto acid.*
   - **CoA** → *PLP (VIT. B_6)*
   - **EX.**
     ALT or GPT
     AST or GOT

2. *Deamination* **:**
   - *Is the removal of amino group from amino acids as ammonia.*
   - *CoA :*
     NAD → B_3
     FAD or FMN → B_2
     PLP → B_6
   - *It's two types :*

     1. *Oxidative :*
        - *L-amino acid. FMN*
        - *D-amino acid. FAD*
        - *L-glutamate dehydrogenase. NAD*
     2. *Non-Oxidative :*
        - *Serine dehydratase.*

3. *Deamidation* :
   Is a removal of amino group of amide as ammonia

4. *Decarboxylation* :
   - *Is removal of a carboxyl group as carbon dioxide CO2 from amino acid.*
   - *CoA → PLP (B_6)*
   - *Ex.*
     ***Histidine** to form **histamine**.*
     ***Tyrosine** to form **tyramine**.*

☆ *Source of Ammonia (NH_3) :*

1. *Deamination of amino acids*
2. *Bacterial action in intestine*
3. *Catabolism of amines*
4. *Catabolism of purines and pyrimidines*

☆ *Fate of Ammonia :*

1. *Produce urea*
2. *Produce non-essential amino acids*
3. *Produce sugar*
4. *Produce purines and pyrimidines*

Transfer the ammonia from tissue to the liver

By 2 amino acids

1. ***Glutamine***
2. ***Alanine***

~~Done by :~~
~~AZZALDEEN Obad~~
~~Abdulrahman Naser~~

Biochemistry
L → 10

Bio Chemistry L-eleven

urea cycle

تعريفها:

Its biochemical reaction converts toxic ammonia to urea in cytosol of mitochondrial in liver every time to remove toxic ammonia.

تحتاج هذه العمليه 4ATP

The cycle of urea converts two amino groups :

One from — Free amino acids (NH4)

And one from — Aspartic acid

And carbon from : CO2
HCO3

Steps of Urea cycle :

Step1: formation of carbamoyl phosphate

HCO3+NH4 ———— carbamoyl phosphate

The enzyme requires is : Caebamoyl phosphate synthetase

The Coenzyme is : N-acetyl glutamate

مهمات:

والا ضروري افعلهم بلون أحمر؟

Step 2: carbamoyl phosphate and ornithine are combined together to form citrulline

carbamoyl phosphate+ornithine ———— citrulline

The enzyme is : ornithine transcarbamoylase

Step 3: citrulline and aspartate to form Argininosuccinate

citrulline+Aspartate ———— Argininosuccinate

The enzyme is : Argininosuccinate synthetase

Step 4: Argininosuccinate is cleaved to arginine and fumarate

Argininosuccinate ———— Arginine + fumarate

The enzyme is : Argininosuccinase (argininosucinate lyase)

Step 5: arginine converted to urea and ornithine

Arginine ———— Urea + ornithine

The enzyme is : Arginase

Urea cycle disorders are characterized by :

- hyperammonemia
- encephalopathy
- vomiting
- lethargy
- mental retardation

- hyperammonemia type I due to deficiency of carbamoyl phosphate synthetase
- hyperammonemia type II due to deficiency of ornithine transcarbamoylase
- citrullinemia due to deficiency of argininosuccinate synthetase
- argininosuccinicaciduria due to deficiency of argininosuccinase
- hyperargininemia due to deficiency of arginase
- N-acetyl glutamate synthetase.

السبب هو نفسه المرض

*All metabolic disorders of urea cycle are autosomal recessive disorders except hyperammonemia type II is X-linked disorders.

Treatment of hyperammonemia :

- dietary protein is restricted
- intravenous sodium phenylacetate and sodium benzoate
- asidification of the intestinal lumen
- antibiotics

Fate of urea :

*the concentration of urea in the plasma is determined by renal function and perfusion.

*the protein content of the diet and the rate of protein catabolism.

*the normal range of blood urea is =< 50 mg/dl.

Uremia : elevated concentration of urea in the blood.

Increased urea levels occur in the following conditions :

- prerenal casuse : 1-congestive heart failure
  2-excessive protein ingestion
  3-gastrointestinal bleeding
  4-shock
  5-dehydration
- renal casuse : 1-renal failure
  2-glomerulonephritis
- postrenal casuse : 1-urinary tract obstruction

Protein turnover :

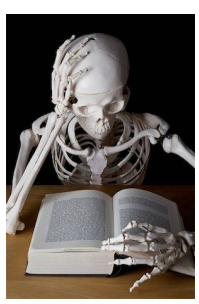
Nitrogen balance : nitrogen intake is equal to nitrogen loss.
Positive Nitrogen balance : nitrogen intake is more than nitrogen loss.
Negative Nitrogen balance : nitrogen intake is less than nitrogen loss.

هذولا الثلاثة المصطلحات حفظ ، زيدوا شوفوا الامثله في الكتاب "١١٢"

There are two main pathways for protein degradation:

- lysosomal-autophagic system
- ubiquitin-proteasome system

تسمع موسيقى وانت تذاكر عادي والا انا بس ؟



Increased blood ereatinine levels occur in the following conditions:

1- Renal disease, acute and chronic renal failure

2-Obstruction of urinary tract

3-Muscular dystrophy

4-Poliomyelitis

5-Congestive heart failure

6-Dehydration

7- Acromegaly and Gigantism

8-Shock

Some genetic disorders of amino acids metabolism

Medical condition	Defective enzyme	Symptoms and effects
Albinism	tyrosinase	White hair , pink skin
Alkaptonuria	Homogentisate 1,2-dioxygenase	Dark pigment in urine; late-developing arthritis
Homocystinuria	Cystathionine B-synthase	Faulty bone, desblesment lens
Maple syrupurine disease	Branched-chain a-keto acid dehydrogenase complex	Vomiting; convulsions; mental retardation , early death
phenylketonuria	Phenylalanine hydroxylase	Vomiting; mental retardation

Ex: albinism is genetic disorders of amino acid metabolism do to deficiency of tyrosinase lead to white hair , pink skin .

Metabolism of nucleotides

معادلات :

1_ DNA DNAase nucleotied nucleotidase nucleosid + phosphat nucleosidase Deoxyribose +A+G+C+T

2_RNA RNAase nucleotied nucleotidase nuclosid + phosphat nucleosidase Ribose +A+G+C+U

Metabolism of purine

1_ De novo (Synthesis of nucleotide from the beginning ،Atom by Atom)

تحتاج وقت اكثر وطاقة اكثر

2_ Salvage (synthesis of nucleotides from the degradation products of nucleic acids)

اسرع وتوفر طاقة فقط نضيف للقاعدة + سكر + فوسفات
خلايا الدم الحمراء تفضل هذي الطريقة لأنها اسهل لها واسرع

Sources of atoms in purine ring

Adenin+Guanin
حلقتين

N9N3 → glutamine
C8C2 → folic acid
N^7C4C5 → glycine
N1 → aspartic acid
C6 → CO^2

✓ يتحول الرايبوز الى دكسي ريبوز بوسطة انزيم Ribonucleotide reductase «--
وهذا يدل على ان ال RNA يصنع قبل DNA

Medical Importance

Chemotherapy : It is a treatment that prevents the growth of cancer cells and normal cells and reduces their growth.

Most affected cells by chemotherapy : GIT , bone marrow stem cells and hair follicles .

Symptoms of chemotherapy : Immune deficiency ، platelet deficiency ، anaemia and hair loss .

Typs of chemotherapy

1_Methoterxate (anti-cancer).
Mechanism →it inhibits dihydrofolate reductase Which transforms folic acid active to inactive .
Used → treatment of leukemia and other types of cancers.

2_Trimethoprim (antibiotic).
Mechanism→ it inhibitor of bacterial dihydrofolate reductase .
Used→ to treat bacterial infections.

3_Azidothymidine.
Mechanism→ it inhibits nucleic acid (RNA) synthesis .
Used→ treatment of AIDS (HIV).

Sources of atoms in pyrimidine ring

حلقة واحدة

N1C4C5C6 → aspartic acid
C2 → CO2
N3 →glutamine

Q. what is end products of purine catabolism?
Uric acid + HN^3

✓ كيف نعلاج النقرص (gout) بعلاج اسمه (Allopurinol) بواسطة تعطيل انزيم (zenthin oxidase) ماهو
وظيفة هذا الانزيم يحول الzenthin الى uric acid
✓ ال purine يسبب النقرص.

Q. what is end products of pyrimidine catabolism?
CO^2,NH^3, B-amino isobutyric acid and B-Ureidopropionase .

Disorders of Purine Metabolism

1_Lesch-Nyhan syndrome.
Symptoms are hyperuricemia , mental retardation, self mutilation and anaemia, renal stones.
which due to complet deficiency of hypoxanthine-guanine phosphoribosyltransferase (HGPRTase)

2_Gout
الأشخاص الذين يتناولون لحوم بكميات كبيرة يترسب ال uric acid في الاقدام لماذا؟ لأنها ابرد منطقة في الجسم وتقلل الذوبانية.
يترسب ال uric acid في سوائل المفاصل ع شكل needl-shape تسمى crystals sodium urate المناعة تهاجمها ويتدمر المفصل ويسبب ال Pain, redness and swelling.

Causes of hyperuricemia

1_Lesch-Nyhan syndrome.
2_Increased activity of phosphorbosyl pyrophosphate synthetase.
3_Von - Gierke's disease
4_Chemotherapy
5_Chronic renal disease
6_Eating a lot of purine - rich diet
Increased tissue catabolism or starvation7_
luekemia8_